Placental abruption: Clinical features and diagnosis

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INTRODUCTION — Placental abruption (also referred to as abruptio placentae) is bleeding at the decidual-placental interface that causes partial or complete placental detachment prior to delivery of the fetus. The diagnosis is typically reserved for pregnancies over 20 weeks of gestation. The major clinical findings are vaginal bleeding and abdominal pain, often accompanied by hypertonic uterine contractions, uterine tenderness, and a nonreassuring fetal heart rate (FHR) pattern.

Abruption is a significant cause of maternal and perinatal morbidity, and perinatal mortality. The perinatal mortality rate is approximately 20-fold higher in comparison to pregnancies without abruption (12 percent versus 0.6 percent, respectively) [1]. The majority of perinatal deaths (up to 77 percent) occur in utero; deaths in the postnatal period are primarily related to preterm delivery [1-4]. However, perinatal mortality associated with abruption appears to be decreasing [1].

INCIDENCE — Placental abruption complicates approximately 1 percent of pregnancies [5,6], with two-thirds classified as severe due to accompanying maternal, fetal, and neonatal morbidity [7]. The incidence appears to be increasing in the United States, Canada, and several Nordic countries [5], possibly due to increases in the prevalence of risk factors for the disorder and/or to changes in case ascertainment [8,9].

In one review, 40 to 60 percent of abruptions occurred before 37 weeks of gestation and 14 percent occurred before 32 weeks [10]. However, gestational age-specific incidence rates vary considerably depending on the etiology [11,12].

PATHOGENESIS AND PATHOPHYSIOLOGY — The immediate cause of the premature placental separation is rupture of maternal vessels in the decidua basalis, where it interfaces with the anchoring villi of the placenta. Rarely, the bleeding originates from the fetal-placental vessels. The accumulating blood splits the decidua, separating a thin layer of decidua with its placental attachment from the uterus. The bleed may be small and self-limited, or may continue to dissect through the placental-decidual interface, leading to complete or near complete placental separation. The detached portion of the placenta is unable to exchange gases and nutrients; when the remaining fetoplacental unit is unable to compensate for this loss of function, the fetus becomes compromised.

The etiology of bleeding at the decidua basalis remains speculative in most cases, despite extensive clinical and epidemiologic research. A small proportion of all abruptions are related to sudden mechanical events, such as blunt abdominal trauma [13] or rapid uterine decompression, which cause shearing of the inelastic placenta due to sudden stretching or contraction of the underlying uterine wall [11,12]. In motor vehicle crashes, an additional factor is rapid acceleration-deceleration of the uterus, which causes uterine stretch without concomitant placental stretch, leading to a shearing force between the placenta and the uterine wall. Although even minor trauma may be associated with an increased risk of preterm birth, severe maternal trauma is associated with a six-fold increased risk of abruption [14].

Uterine abnormalities, cocaine use, and smoking are additional less common causes of abruption. Uterine anomalies (eg, bicornuate uterus), uterine synechiae, and leiomyoma are mechanically and biologically unstable sites for placental implantation; abruption at these sites may be due to inadequate decidualization and/or shear. Suboptimal trophoblastic implantation may also explain the increased risk of abruption among women with a prior cesarean (odds ratio [OR] 2.3, 95% CI 1.5-3.6) [15]. The pathophysiological effect of cocaine in the genesis of abruption is unknown, but may be related to cocaine-induced vasoconstriction leading to ischemia, reflex vasodilatation, and disruption of vascular integrity. As many as 10 percent of women using cocaine in the third trimester will develop placental abruption [16-18]. The mechanism(s) that underlie the relationship between smoking cigarettes and abruption also remains unclear. One hypothesis is that the vasoconstrictive effects of smoking cause placental hypoperfusion, which could result in decidual ischemia, necrosis, and hemorrhage leading to premature placental separation [19,20].

Most abruptions appear to be related to a chronic placental disease process. In these cases, abnormalities in the early development of the spiral arteries lead to decidual necrosis, placental inflammation and possibly infarction, and ultimately vascular disruption and bleeding [11,12,21-23]. High pressure arterial hemorrhage in the central area of the placenta leads to rapid development of potentially life-threatening clinical manifestations of abruption (eg, severe bleeding, maternal disseminated intravascular coagulation, fetal heart rate [FHR] abnormalities). Low pressure venous hemorrhage, typically at the periphery of the placenta (marginal abruption), is more likely to result in clinical manifestations that occur over time (eg, light

intermittent bleeding, oligohydramnios, and fetal growth restriction associated with redistribution of cerebral blood flow [decrease in the middle cerebral artery pulsatility index [24]]).

Thrombin plays a key role in the clinical consequences of placental abruption, and may be important in its pathogenesis, as well. It is formed via two pathways: in one pathway, decidual bleeding leads to release of tissue factor (thromboplastin) from decidual cells, which generates thrombin [25]. In the other pathway, decidual hypoxia induces production of vascular endothelial growth factor (VEGF), which acts directly on decidual endothelial cells to induce aberrant expression of tissue factor, which then generates thrombin [26]. The production of thrombin can lead to the following clinical sequelae:

- Uterine hypertonus and contractions, as thrombin is a potent, direct uterotonic agent [27].
- Enhanced expression of matrix metalloproteinases [25,28], up-regulation of genes involved in apoptosis [26], and induced expression of inflammatory cytokines (predominantly interleukin-8), leading to tissue necrosis and degradation of extracellular matrix [26,29,30]. A vicious cycle then ensues, resulting in further vascular disruption, and often leading to initiation of labor and rupture of membranes (algorithm 1).

  In women with premature rupture of membranes, the risk of placental abruption increases with increasing latency, which suggests that inflammation subsequent to membrane rupture can induce rather than result from the cascade of events leading to placental separation [31-35].
- Triggering of coagulation. If a massive amount of tissue factor (thromboplastin) is released, a massive amount of thrombin is generated and enters the maternal circulation over a brief period of time [36]. This overwhelms hemostatic control mechanisms, without allowing sufficient time for recovery of compensatory mechanisms. The clinical consequence is a profound systemic bleeding diathesis and, due to widespread intravascular fibrin deposition, ischemic tissue injury and microangiopathic hemolytic anemia (ie, disseminated intravascular coagulation [DIC]).
- Functional progesterone withdrawal by reduced expression of progesterone receptors in decidual cells, which initiates or contributes to uterine contractility [37].

CLINICAL FEATURES

Risk factors — Previous abruption is the strongest risk factor for abruption, with recurrence risks of 10- to 15-fold higher [38], and as high as 93-fold higher (95% CI 62-139) [6]. Other major risk factors in singleton and twin gestations are described in the table (table 1) [39]. Smoking is one of the few modifiable risk factors for abruption: it is associated with a 2.5-fold increased risk of abruption severe enough to result in fetal death and the risk increases by 40 percent for each pack per day smoked [40]. The combination of cigarette smoking and hypertension has a synergistic effect on risk of abruption [41]. Hypertensive women have a five-fold increased risk of severe abruption compared to normotensive women, and antihypertensive therapy does not appear to reduce the risk of placental abruption among women with chronic hypertension [42]. Vitamin C-E supplementation appeared to mitigate the risk of abruption among smokers in one study [43], but this finding must be cautiously interpreted given the small number of events and wide confidence interval.

A modest increase in the risk of abruption has also been noted in women with asthma (adjusted OR 1.22, 95% CI 1.09-1.36) [44]. The sisters of women who have had an abruption appear to be at increased risk of also having an abruption (odds ratio 1.7 to 2.1) [45]. A case-control study noted that pregnancies complicated by abruption were almost twice as likely to be associated with major fetal congenital anomalies than pregnancies without abruption (OR 1.92, 95% CI 1.6-2.52), especially when the fetus was growth restricted [46].

Evidence of an association between elevated thyroperoxidase antibodies and hypothyroidism in relation to abruption has also been reported. For example, one study reported that placental abruption was present more often among women with subclinical hypothyroidism compared with euthyroid controls [47]. However, most women with abruption are not affected, these diagnoses are not highly predictive of abruption, and there is no evidence that treatment of asymptomatic patients will reduce the risk of abruption. (See "Hypothyroidism during pregnancy: Clinical manifestations, diagnosis, and treatment", section on 'Pregnancy complications'.)

Acute abruption

Patient presentation — Women with an acute abruption classically present with the abrupt onset of vaginal bleeding, mild to moderate abdominal and/or back pain, and uterine contractions. Back pain is prominent when the placenta is on the posterior wall of the uterus. The uterus is often firm, and may be rigid and tender.

Contractions are usually high frequency and low amplitude, but a contraction pattern typical of labor is also possible and labor may proceed rapidly.

Vaginal bleeding ranges from mild and clinically insignificant to severe and life-threatening. Blood loss may be underestimated because bleeding may be retained behind the placenta and thus difficult to quantify. The amount of vaginal bleeding correlates poorly with the degree of placental separation and does not serve as a useful marker of impending fetal or maternal risk. Abdominal pain is a better predictor of poor outcome [48]. Maternal hypotension and fetal heart rate (FHR) abnormalities

suggest clinically significant separation that could result in fetal death and severe maternal morbidity. When placental separation exceeds 50 percent, acute disseminated intravascular coagulation and fetal death are common [49,50].

In 10 to 20 percent of placental abruptions, patients present with only preterm labor, and no or scant vaginal bleeding. In these cases, termed "concealed abruption," all or most of the blood is trapped between the fetal membranes and decidua, rather than escaping through the cervix and vagina [49]. Therefore, in pregnant women with abdominal pain and uterine contractions, even a small amount of vaginal bleeding should prompt close maternal and fetal evaluation for placental abruption. In other cases, a small concealed abruption may be asymptomatic and only recognized as an incidental finding on an ultrasound.

Occasionally, the signs and symptoms of abruption develop after rapid uterine decompression, such as after uncontrolled rupture of membranes in the setting of polyhydramnios or after delivery of a first twin. Signs and symptoms of abruption also may occur after maternal abdominal trauma or a motor vehicle crash. In these cases, placental abruption generally presents within 24 hours of the precipitating event and tends to be severe. The clinical presentation and obstetrical evaluation of pregnant trauma victims are described in detail separately. (See "Initial evaluation and management of pregnant women with major trauma", section on 'Initial evaluation and management of major trauma'.)

Laboratory findings — The degree of maternal hemorrhage correlates with the degree of hematological abnormality; fibrinogen levels have the best correlation with severity of bleeding [51], overt disseminated intravascular coagulation, and the need for transfusion of multiple blood products [52]. Initial fibrinogen values of ≤200 mg/dL are reported to have 100 percent positive predictive value for severe postpartum hemorrhage, while levels of ≥400 mg/dL have a negative predictive value of 79 percent [53]. Mild separation/hemorrhage may not be associated with any abnormalities of commonly used tests of hemostasis.

Severe abruption can lead to disseminated intravascular coagulation (DIC). DIC occurs in 10 to 20 percent of severe abruptions with death of the fetus. (See "Clinical features, diagnosis, and treatment of disseminated intravascular coagulation in adults", section on 'Diagnostic evaluation'.)

The diagnosis of acute DIC is confirmed by demonstrating increased thrombin generation (eg, decreased fibrinogen) and increased fibrinolysis (eg, elevated fibrin degradation products [FDPs] and D-dimer). However, laboratory findings suggestive of mild DIC need to be interpreted with caution in pregnancy because of normal pregnancy-related increases in the concentration of almost all coagulation factors and a normal mild decrease in platelet count. (See "Hematologic changes in pregnancy".)

The Kleihauer-Betke test is positive in a small proportion of abruptions. There is poor correlation between the results of this test and the presence or absence of abruption [49,54-56] with a sensitivity of only 4 percent [57].

Even prior to clinical or sonographic findings of abruption, there may be early markers of ischemic placental disease detected during routine prenatal care. Abnormalities of maternal serum aneuploidy analytes (eg, increased alpha fetoprotein or human chorionic gonadotropin, decreased pregnancy-associated plasma protein A or unconjugated estriol not explained by fetal abnormalities) carry up to a 10-fold risk of subsequent abruption [58-63]. Data also suggest that a low fetal fraction identified during first-trimester cell-free fetal DNA aneuploidy screening has a 2.5-fold increased risk of composite obstetrical morbidity, including placental abruption [64]. Ongoing investigation on early maternal serum metabolomics and placenta-specific microRNAs may yield information on the biomarkers of placental abruption [65,66].

Imaging — Identification of a retroplacental hematoma is the classic ultrasound finding of placental abruption (image 1). Retroplacental hematomas have a variable appearance; they can appear solid, complex, and hypo-, hyper-, or iso-echoic compared to the placenta. Hypoechogenicity and sonolucency are features of resolving rather than acute hematomas (image 2).

Sonographic findings consistent with placental abruption are associated with the worse maternal and perinatal outcomes. However, whether a hematoma is identified depends on the extent of hemorrhage, chronicity of the bleeding, and extent that blood has escaped through the cervix (image 3A-B and image 4A-B). Although the worst outcomes appear to occur when there is sonographic evidence of a retroplacental hematoma [67,68], the absence of retroplacental hematoma does not exclude the possibility of severe abruption because blood may not collect behind the uterus. The sensitivity of ultrasound findings for diagnosis of abruption is only 25 to 60 percent [68-71], but the positive predictive value is high (88 percent) when ultrasound findings suggestive of abruption are present [68,69,72].

A thorough search for other findings in symptomatic patients may improve the sensitivity and specificity of ultrasound. These findings include subchorionic collections of fluid (even remote from the placental attachment site), echogenic debris in the amniotic fluid, or a thickened placenta, especially if it shimmers with maternal movement ("Jello" sign) [72].

Magnetic resonance imaging can detect abruptions missed by ultrasound examination, but increased diagnostic certainty is unlikely to change management or be cost-effective [73,74]. Although contrast enhanced computed tomography is rarely used as a first-line imaging test in pregnancy, it may be performed after maternal trauma to rule out internal injury. In this setting, it has high sensitivity but low specificity for identifying placental abruption [75] and can estimate the extent (<25 percent, 25 to 50 percent, >50 percent) of placental separation.

Consequences — For the mother, the potential consequences of abruption are primarily related to the severity of the placental separation, while the risks to the fetus are related to both the severity of the separation and the gestational age at which delivery occurs [2,11,12,49,50,76-80]. With mild placental separation, there may be no significant adverse effects. As the degree of placental separation increases, the maternal and perinatal risks also increase [2,49,50,76,81]. In a retrospective cohort study, the frequency of serious maternal complications among women with no abruption, mild abruption, and severe abruption was 15, 33, and 142 per 10,000 women, respectively [7].

Maternal:

- Excessive blood loss and DIC generally necessitate blood transfusion and can lead to hypovolemic shock, renal failure, adult respiratory distress syndrome, multiorgan failure, peripartum hysterectomy and, rarely, death [10,49].
- Emergency cesarean delivery for fetal or maternal indications
- In addition to acute complications, placental syndromes, including abruption, have been associated with increased risk of premature cardiovascular disease and doubling of the risk of death after coronary artery revascularization [82-84].

Fetal and neonatal:

- Perinatal morbidity and mortality related to hypoxemia, asphyxia, low birth weight, and/or preterm delivery [1,2,50,76-78,81,85,86].
- Fetal growth restriction (with chronic abruption) (see 'Chronic abruption' below) [2,50,76,77,81,87].

In population-based studies, the perinatal mortality rate is about 12 percent (versus 0.6 percent in births without abruption) [1-4]. More than 50 percent of abruption-related perinatal deaths are stillborns due to intrauterine asphyxia, which generally occurs when over 45 percent of the placenta detaches, particularly when central in location [10,88]. Placental abruption is implicated in up to 10 percent of preterm births and perinatal death is related to preterm delivery in at least 30 percent of cases [49,50,81]. Preterm birth may be iatrogenic due to the nonreassuring fetal or maternal condition, or it may be related to preterm labor or preterm premature rupture of membranes [49]. In women admitted with the diagnosis of abruption, perinatal mortality is independently associated with preterm delivery (especially less than 30 weeks of gestation) and abdominal trauma [89].

Fetal asphyxia, preterm birth, and growth restriction can be associated with short- and long-term sequelae. A study of 29 neonates from pregnancies complicated by abruption at a median of 29 weeks of gestation reported a 10-fold increase in periventricular leukomalacia compared with matched neonates in pregnancies without abruption; the rate of periventricular leukomalacia was 34 percent [90]. Long-term neurodevelopmental deficits among children born after placental abruption appear to be mediated largely through preterm delivery [91]. (See "Short-term complications of the preterm infant" and "Systemic effects of perinatal asphyxia" and "Infants with fetal (intrauterine) growth restriction" and "Long-term complications of the preterm infant".)

Placental pathology — In one large series, gross examination of the placenta at delivery revealed the following estimated frequencies of placental separation: less than 25 percent placental separation (54 percent); 25 to 49 percent placental separation (16 percent); 50 to 74 percent placental separation (13 percent); over 75 percent placental separation (17 percent) [50].

Histopathological examination of the placenta shortly after an acute abruption may not reveal any abnormality. In less acute cases, an organizing retroplacental hematoma indenting the parenchyma may be noted [22]. Recent infarcts may be present and are characterized by preservation of villous stromal architecture, eosinophilic degeneration of the syncytiotrophoblast, and villous agglutination with scattered intervillous neutrophils. These infarcts take approximately four to six hours to develop.

Histological findings, such as diffuse retromembranous and/or intradecidual hemorrhages, pigmented histiocytes, irregular basal intervillous thrombi, and recent villous stromal hemorrhage, are not specific for the diagnosis. Histologic evidence of decidual hemorrhage is noted in 2 to 4 percent of deliveries; most cases are associated with preterm premature rupture of membranes or preterm labor and delivery rather than a clinical diagnosis of abruption [49,92].

Recurrence — Women with placental abruption are at several-fold higher risk of abruption and other manifestations of ischemic placental disease in a subsequent pregnancy. (See "Placental abruption: Management" and "Placental abruption: Management", section on 'Recurrence risk' and "Placental abruption: Management", section on 'Other risks'.)

Chronic abruption — Women with chronic abruption experience relatively light, chronic, intermittent bleeding and clinical manifestations of ischemic placental disease that develop over time, such as oligohydramnios (termed chronic abruption –oligohydramnios sequence [93]), fetal growth restriction, and preeclampsia [32,33,87]. They are also at risk of preterm premature rupture of membranes.

Coagulation studies are usually normal. Ultrasound examination may identify a placental hematoma (retromembranous, marginal, or central), and serial examination may reveal fetal growth restriction and/or oligohydramnios. Abruption in the

second trimester accompanied by oligohydramnios has a dismal prognosis, including high rates of fetal death, preterm birth, and serious neonatal morbidity or death [93-96].

Histological examination of the placenta may show chronic lesions, such as chronic deciduitis (lymphocytes with or without plasma cells), decidual necrosis, villitis, decidual vasculopathy (specifically, in the vessels of the extraplacental membrane roll), placental infarction, intervillous thrombosis, villous maldevelopment, and hemosiderin deposition [22].

DIAGNOSIS — The diagnosis of abruptio placentae is primarily clinical, but findings from imaging, laboratory, and postpartum pathologic studies can be used to support the clinical diagnosis. Women with an acute abruption classically present with the abrupt onset of mild to moderate vaginal bleeding and abdominal and/or back pain, accompanied by uterine contractions. The uterus has increased tone/rigidity and may be tender both during and between contractions. In patients with classic symptoms, fetal heart rate (FHR) abnormalities or intrauterine fetal demise and/or disseminated intravascular coagulation strongly support the clinical diagnosis and indicate extensive placental separation.

Ultrasound examination is useful for identifying a retroplacental hematoma and for excluding other disorders associated with vaginal bleeding and abdominal pain (see 'Differential diagnosis' below). A retroplacental hematoma is the classic ultrasound finding and strongly supports the clinical diagnosis, but is absent in many patients with abruption.

Postpartum, the absence of characteristic placental findings does not exclude the diagnosis. In a multicenter case-control study, standardized gross and histopathological evaluation of the placenta was only able to confirm a strong clinical diagnosis in 30 percent of cases (49/162) [22].

DIFFERENTIAL DIAGNOSIS — In pregnant women with suspected abruption, the differential diagnosis of vaginal bleeding accompanied by pain and contractions includes labor, placenta previa, uterine rupture, and subchorionic hematoma.

The signs and symptoms of labor have a more gradual onset than those of abruption. The onset of labor (preterm or term) is characterized by mild uterine contractions at infrequent and/or irregular intervals; the contractions become more regular and painful over time and are accompanied by cervical dilation and/or effacement. Mucus that has accumulated in the cervix may be discharged as clear, pink, or slightly bloody secretions (ie, mucus plug, bloody show), sometimes several days before labor begins. Early labor is usually associated with less bleeding, less uterine rigidity, less abdominal pain, and fewer high frequency contractions compared with abruption; however, there is an overlap in symptoms since abruption may trigger labor. (See "Diagnosis of preterm labor and overview of preterm birth".)

The characteristic clinical presentation of placenta previa is painless vaginal bleeding after 20 weeks of gestation; however, 10 to 20 percent of women present with uterine contractions associated with the bleeding. Thus, abruption and placenta previa can be difficult to distinguish clinically since abruption may not be associated with significant pain and placenta previa may not be painless. In pregnant women with vaginal bleeding, an ultrasound examination should be performed to determine whether placenta previa is the source. (See "Clinical features, diagnosis, and course of placenta previa".)

Uterine rupture is most common in women with a prior hysterotomy. Signs of uterine rupture may include the sudden onset fetal heart rate (FHR) abnormalities, vaginal bleeding, constant abdominal pain, cessation of uterine contractions, recession of the presenting part, and maternal hypotension and tachycardia. Many of these symptoms are common to abruption because uterine rupture often leads to placental separation. (See "Rupture of the unscarred uterus" and "Uterine rupture after previous cesarean delivery".)

Subchorionic hematoma is believed to result from partial detachment of the chorionic membranes from the uterine wall, in contrast to abruption, which is due to detachment of the placenta from the uterine wall [97]. Patients are asymptomatic or experience light vaginal bleeding. In contrast to abruption, abdominal pain is typically absent, a minority of patients experience cramping or contractions, and the diagnosis is usually made before rather than after 20 weeks of gestation [98]. The diagnosis is based on ultrasound findings of a hypoechoic or anechoic crescent-shape area behind the fetal membranes, which may also elevate the edge of the placenta [99]. Women with subchorionic hematomas have a greater than five-fold increased risk of developing an abruption, as well as other pregnancy complications (eg, preterm labor, premature rupture of membranes) [99].

DETERMINING THE CAUSE OF ABRUPTION — The cause of most abruptions cannot be determined with certainty. A thorough history and physical examination may identify risk factors for the disorder, such as smoking, hypertension/preeclampsia, recent cocaine use, trauma, rapid uterine decompression, premature rupture of membranes, chorioamnionitis, or a prior history of abruption, preeclampsia, or delivery of a small for gestational age neonate.

Although use of cocaine can result in abruption, we do not routinely perform toxicology screening on all patients with unexplained placental abruption. We obtain urine toxicology tests in patients with risk factors for substance use [100]. (See "Clinical assessment of substance use disorders" and "Substance misuse in pregnant women".)

Laboratory screening for underlying conditions, such as subclinical hypothyroidism, at the time of presentation is not recommended, as it provides little assistance in the acute assessment or management of abruption.

MANAGEMENT — (See "Placental abruption: Management".)

INFORMATION FOR PATIENTS — UpToDate offers two types of patient education materials, “The Basics” and “Beyond the Basics.” The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on “patient info” and the keyword (s) of interest.)

- Basics topic (see "Patient education: Placental abruption (The Basics)")

SUMMARY AND RECOMMENDATIONS

- Placental abruption refers to partial or complete separation of the placenta prior to delivery of the fetus. (See 'Introduction' above.)
- Most placental abruptions are related to a chronic pathologic vascular process, but some are due to acute events, such as trauma or vasoconstriction. The immediate cause of placental separation is rupture of maternal blood vessels in the decidua basalis. The subsequent release of tissue factor and generation of thrombin lead to many of the clinical sequelae of acute abruption. (See 'Pathogenesis and pathophysiology' above.)
- Risk factors for placental abruption are listed in the table (table 1). (See 'Risk factors' above.)
- The classic symptoms and signs of acute placental abruption are vaginal bleeding, abdominal pain, contractions, uterine rigidity and tenderness, and possibly a nonreassuring fetal heart rate (FHR) tracing. In 10 to 20 percent of placental abruptions, patients present with only preterm labor, and no vaginal bleeding. Some abruptions are asymptomatic. The amount of bleeding does not correlate well with the extent of maternal hemorrhage and cannot be used as a marker to gauge the severity of premature placental separation. FHR abnormalities suggest clinically significant separation that could result in fetal death. (See 'Patient presentation' above.)
- A retroplacental clot is the classic ultrasound finding of placental abruption, but is not always present. (See 'Imaging' above.)
- When placental separation exceeds 50 percent, acute disseminated intravascular coagulation and fetal death are common. (See 'Laboratory findings' above and 'Consequences' above.)
- In contrast to acute abruption, patients with chronic placental abruption experience relatively light, chronic, intermittent bleeding and exhibit clinical manifestations that develop over time, such as oligohydramnios, fetal growth restriction, and preeclampsia. (See 'Chronic abruption' above.)
- The diagnosis of acute abruption is clinical and based on the abrupt onset of mild to moderate vaginal bleeding and abdominal and/or back pain, accompanied by uterine contractions. A retroplacental clot is the classic ultrasound finding and strongly supports the clinical diagnosis, but is absent in many patients with abruption. In patients with classic symptoms, FHR abnormalities or intrauterine fetal demise and/or disseminated intravascular coagulation strongly support the clinical diagnosis and indicate extensive placental separation. (See 'Diagnosis' above.)
- Women with placental abruption are at several-fold higher risk of abruption in a subsequent pregnancy, especially if the abruption was severe. (See 'Recurrence' above.)

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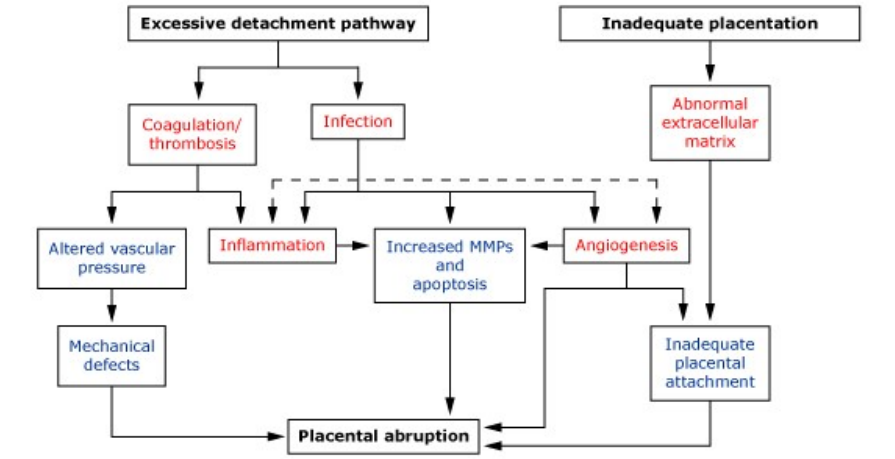
Topic 6826 Version 30.0

GRAPHICS

Conceptual model showing coagulation/thrombosis, infection, inflammation, angiogenesis, and collagen metabolism pathways to placental abruption



MMP = matrix metalloproteinases

Graphic 54747 Version 2.0

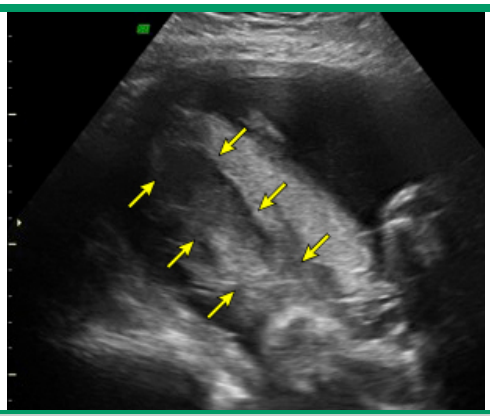
Major risk factors for placental abruption

Risk factor	Singleton gestations		Twin gestations	
	Strength	RR/OR	Strength	RR/OR
Acute etiology				
Abdominal trauma/accidents	+++			
Cocaine or other drug abuse	+++	5.0-10.0		
Polyhydramnios	++	2.0-3.0	++	1.7
Obstetrical/medical risk factors				
Chronic hypertension	++	1.8-5.1		
Preeclampsia/Pregnancy induced hypertension	++	0.4-4.5		
Eclampsia	+++	3.0-5.5	++	1.6-2.0
Premature rupture of membranes	++	1.8-5.1	++	1.5-2.5
Chorioamnionitis	++	2.0-2.5	++	1.7
Previous ischemic placental disease				
Preeclampsia	++	1.5		
Fetal growth restriction/Small for gestation age infant	++	1.4		
Previous abruption	++++	8.0-12.0		
Sociodemographic/behavioral				
Maternal age	+	1.1-1.3	+	1.1-1.4
Parity	+	1.1-1.6	+	1.1-1.7
Smoking during pregnancy	++	1.4-2.5	++	1.7
Male infant sex	+/-	0.9-1.3		

OR: odds ratio; RR: relative risk.

Graphic 75801 Version 3.0

Ultrasound image retroplacental hematoma

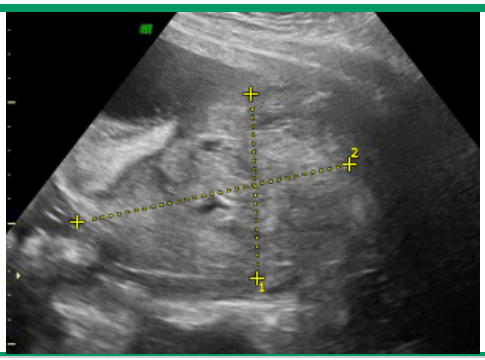


Posterior placenta with large retroplacental hematoma outlined by arrows.

Courtesy of Martin R Chavez, MD.

Graphic 83126 Version 1.0

Organizing intrauterine hematoma

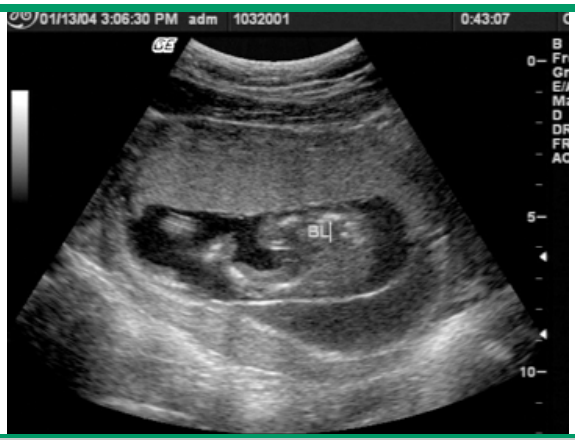


Posterior heterogeneous collection (delineated by + signs) in the lower uterine segment representing an organized clot measuring 7 x 12 cm.

Courtesy of Martin R Chavez, MD.

Graphic 83127 Version 1.0

Retromembranous bleed 1

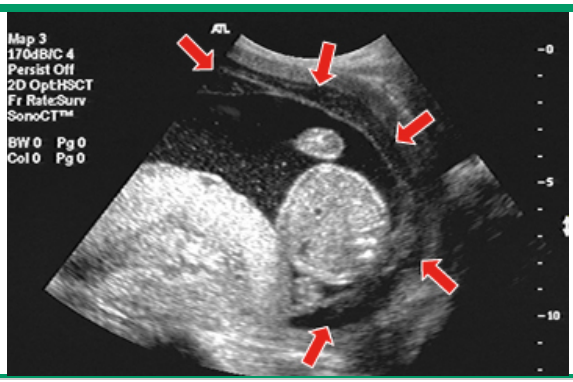


A large retromembranous bleed is present posteriorly.

Courtesy of Charles Lockwood, MD.

Graphic 72314 Version 2.0

Retromembranous bleed 2

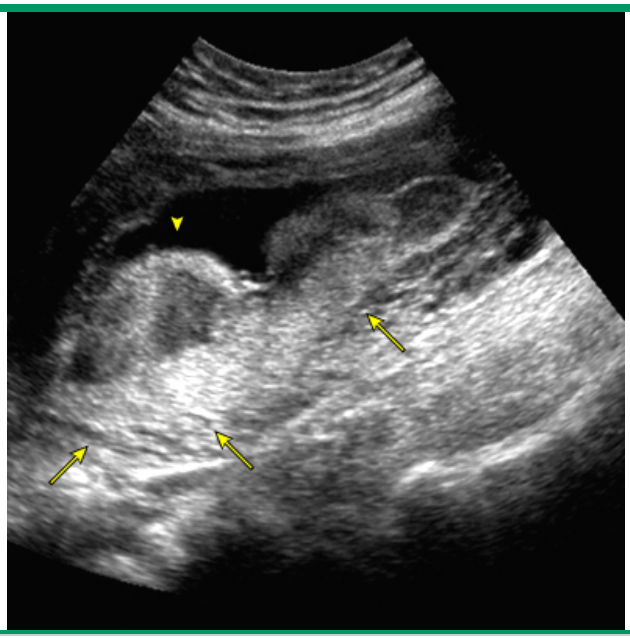


Arrows point to a retromembranous bleed.

Courtesy of Charles Lockwood, MD.

Graphic 51286 Version 3.0

Preplacental hematoma

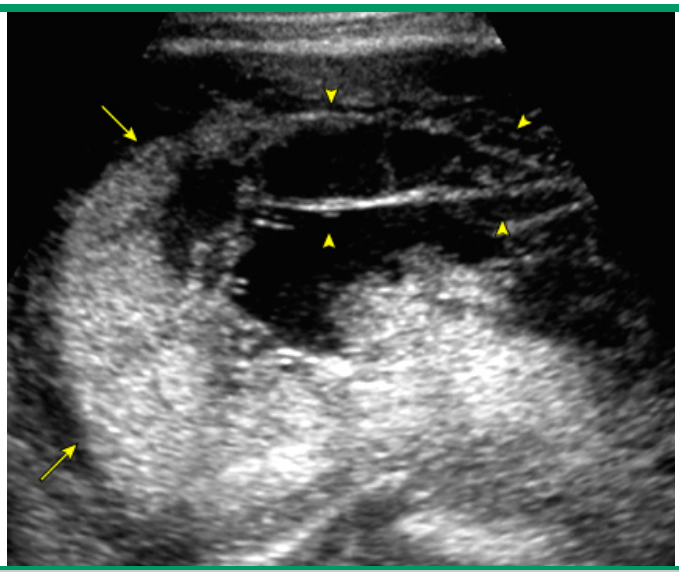


Posterior placenta (arrows) with preplacental hematoma (arrowhead).

Courtesy of Wendy L Kinzler, MD.

Graphic 69163 Version 3.0

Subchorionic hematoma



Posterior fundal placenta (arrows) with an anterior organized subchorionic hematoma (arrowheads) arising from the superior edge of the placenta.

Courtesy of Wendy L Kinzler, MD.

Graphic 79004 Version 3.0

Contributor Disclosures

Cande V Ananth, PhD, MPH Nothing to disclose **Wendy L Kinzler, MD** Nothing to disclose **Charles J Lockwood, MD, MHCM** Consultant/Advisory Boards: Celula [Aneuploidy screening (No current products or drugs in the US)]. **Vanessa A Barss, MD, FACOG** Nothing to disclose

Contributor disclosures are reviewed for conflicts of interest by the editorial group. When found, these are addressed by vetting through a multi-level review process, and through requirements for references to be provided to support the content. Appropriately referenced content is required of all authors and must conform to UpToDate standards of evidence.

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